UNIT TERMINAL OBJECTIVE

5-4 At the completion of this unit, the paramedic student will be able to integrate pathophysiological principles and assessment findings to formulate a field impression and implement a treatment plan for the patient with an endocrine problem.

COGNITIVE OBJECTIVE

At the completion of this unit, the paramedic student will be able to:

5-4.1 Describe the incidence, morbidity and mortality of endocrinologic emergencies. (C-1)
5-4.2 Identify the risk factors most predisposing to endocrinologic disease. (C-1)
5-4.3 Discuss the anatomy and physiology of organs and structures related to endocrinologic diseases. (C-1)
5-4.4 Review the pathophysiology of endocrinologic emergencies. (C-1)
5-4.5 Discuss the general assessment findings associated with endocrinologic emergencies. (C-1)
5-4.6 Identify the need for rapid intervention of the patient with endocrinologic emergencies. (C-1)
5-4.7 Discuss the management of endocrinologic emergencies. (C-1)
5-4.8 Describe osmotic diuresis and its relationship to diabetes. (C-1)
5-4.9 Describe the pathophysiology of adult onset diabetes mellitus. (C-1)
5-4.10 Describe the pathophysiology of juvenile onset diabetes mellitus. (C-1)
5-4.11 Describe the effects of decreased levels of insulin on the body. (C-1)
5-4.12 Correlate abnormal findings in assessment with clinical significance in the patient with a diabetic emergency. (C-3)
5-4.13 Discuss the management of diabetic emergencies. (C-1)
5-4.14 Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient with a diabetic emergency. (C-3)
5-4.15 Differentiate between the pathophysiology of normal glucose metabolism and diabetic glucose metabolism. (C-3)
5-4.16 Describe the mechanism of ketone body formation and its relationship to ketoacidosis. (C-1)
5-4.17 Discuss the physiology of the excretion of potassium and ketone bodies by the kidneys. (C-1)
5-4.18 Describe the relationship of insulin to serum glucose levels. (C-1)
5-4.19 Describe the effects of decreased levels of insulin on the body. (C-1)
5-4.20 Describe the effects of increased serum glucose levels on the body. (C-1)
5-4.21 Discuss the pathophysiology of hypoglycemia. (C-1)
5-4.22 Discuss the utilization of glycogen by the human body as it relates to the pathophysiology of hypoglycemia. (C-3)
5-4.23 Describe the actions of epinephrine as it relates to the pathophysiology of hypoglycemia. (C-3)
5-4.24 Recognize the signs and symptoms of the patient with hypoglycemia. (C-1)
5-4.25 Describe the compensatory mechanisms utilized by the body to promote homeostasis relative to hypoglycemia. (C-1)
5-4.26 Describe the management of a responsive hypoglycemic patient. (C-1)
5-4.27 Correlate abnormal findings in assessment with clinical significance in the patient with hypoglycemia. (C-1)
5-4.28 Discuss the management of the hypoglycemic patient. (C-1)
5-4.29 Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient with hypoglycemia. (C-3)
5-4.30 Discuss the pathophysiology of hyperglycemia. (C-1)
5-4.31 Recognize the signs and symptoms of the patient with hyperglycemia. (C-1)
5-4.32 Describe the management of hyperglycemia. (C-1)
5-4.33 Correlate abnormal findings in assessment with clinical significance in the patient with hyperglycemia. (C-3)
5-4.34 Discuss the management of the patient with hyperglycemia. (C-1)
5-4.35 Integrate the pathophysiological principles and the assessment findings to formulate a field impression

and implement a treatment plan for the patient with hyperglycemia. (C-3)
5-4.36 Discuss the pathophysiology of nonketotic hyperosmolar coma. (C-1)
5-4.37 Recognize the signs and symptoms of the patient with nonketotic hyperosmolar coma. (C-1)
5-4.38 Describe the management of nonketotic hyperosmolar coma. (C-1)
5-4.39 Correlate abnormal findings in assessment with clinical significance in the patient with nonketotic hyperosmolar coma. (C-3)
5-4.40 Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient with nonketotic hyperosmolar coma. (C-3)
5-4.41 Discuss the management of the patient with hyperglycemia. (C-1)
5-4.42 Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient with hyperglycemia. (C-3)
5-4.43 Discuss the pathophysiology of diabetic ketoacidosis. (C-1)
5-4.44 Recognize the signs and symptoms of the patient with diabetic ketoacidosis. (C-1)
5-4.45 Describe the management of diabetic ketoacidosis. (C-1)
5-4.46 Correlate abnormal findings in assessment with clinical significance in the patient with diabetic ketoacidosis. (C-3)
5-4.47 Discuss the management of the patient with diabetic ketoacidosis. (C-1)
5-4.48 Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient with diabetic ketoacidosis. (C-3)
5-4.49 Discuss the pathophysiology of thyrotoxicosis. (C-1)
5-4.50 Recognize signs and symptoms of the patient with thyrotoxicosis. (C-1)
5-4.51 Describe the management of thyrotoxicosis. (C-1)
5-4.52 Correlate abnormal findings in assessment with clinical significance in the patient with thyrotoxicosis. (C-3)
5-4.53 Discuss the management of the patient with thyrotoxicosis. (C-1)
5-4.54 Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient with thyrotoxicosis. (C-3)
5-4.55 Discuss the pathophysiology of myxedema. (C-1)
5-4.56 Recognize signs and symptoms of the patient with myxedema. (C-1)
5-4.57 Describe the management of myxedema. (C-1)
5-4.58 Correlate abnormal findings in assessment with clinical significance in the patient with myxedema. (C-3)
5-4.59 Discuss the management of the patient with myxedema. (C-1)
5-4.60 Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient with myxedema. (C-3)
5-4.61 Discuss the pathophysiology of Cushing's syndrome. (C-1)
5-4.62 Recognize signs and symptoms of the patient with Cushing's syndrome. (C-1)
5-4.63 Describe the management of Cushing's syndrome. (C-1)
5-4.64 Correlate abnormal findings in assessment with clinical significance in the patient with Cushing's syndrome. (C-3)
5-4.65 Discuss the management of the patient with Cushing’s syndrome. (C-1)
5-4.66 Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient with Cushing's syndrome. (C-3)
5-4.67 Discuss the pathophysiology of adrenal Insufficiency. (C-1)
5-4.68 Recognize signs and symptoms of the patient with adrenal insufficiency. (C-1)
5-4.69 Describe the management of adrenal insufficiency. (C-1)
5-4.70 Correlate abnormal findings in assessment with clinical significance in the patient with adrenal insufficiency. (C-3)
5-4.71 Discuss the management of the patient with adrenal insufficiency. (C-1)
5-4.72 Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient with adrenal insufficiency. (C-3)
5-4.73 Integrate the pathophysiological principles to the assessment of a patient with a endocrinological

emergency. (C-3)
5-4.74 Differentiate between endocrine emergencies based on assessment and history. (C-3)
5-4.75 Correlate abnormal findings in the assessment with clinical significance in the patient with endocrinologic emergencies. (C-3)
5-4.76 Develop a patient management plan based on field impression in the patient with an endocrinologic emergency. (C-3)

AFFECTIVE OBJECTIVES
None identified for this unit.

PSYCHOMOTOR OBJECTIVES
None identified for this unit.

DECLARATIVE

I. Introduction
  A. Epidemiology
    1. Incidence
    2. Mortality/ morbidity
    3. Risk factors
    4. Prevention strategies
  B. Anatomy and physiology

II. General pathophysiology, assessment and management
  A. Pathophysiology
    1. Endocrine system
      a. Integrated chemical and coordination system enabling
        (1) Reproduction
        (2) Growth and development
        (3) Regulation of energy
      b. Works with the nervous system to help
        (1) Maintain an internal homeostasis of the body
        (2) Coordinate responses to environmental changes and stress
      c. Composed of glands or glandular tissue that synthesize, store and secrete chemical messengers (hormones) that affect specific target organs and body tissues
      d. Specificity of this system is determined by the affinity of receptors on target organs and body tissues to a particular hormone
    2. Endocrine glands
      a. Ductless glands
        (1) Highly vascular
        (2) Synthesize and secrete hormones
        (3) Specific glands
          (a) Hypothalamus
          (b) Pituitary
          (c) Thyroid
          (d) Parathyroid
          (e) Adrenal
          (f) Kidneys
          (g) Pancreatic islets
          (h) Ovaries
          (i) Testes
          (j) Hormones
        (4) Common characteristics
          (a) Circulation through the blood
          (b) Secretion of minute but effective amounts at predictable but variable intervals bind to specific cellular receptors to change intercellular metabolism
        (5) Structure
  B. Assessment findings
    1. Scene size-up
      a. Scene safety
      b. Personal protective equipment (PPE)

(1) General impression
(2) Trauma
(a) Responsive
(b) Unresponsive
(3) Medical
(a) Responsive
(b) Unresponsive

c. Nature of illness

2. Initial assessment
a. Airway
b. Breathing
c. Circulation
d. Disability
e. Chief complaint

3. Focused history
a. Onset
b. Provoking factors
c. Time
d. Nausea/ vomiting
e. Weight loss
f. Last meal
g. Non-specific
h. Changes in
(1) Energy level
(2) Alertness
(3) Sleep patterns
(4) Mood
(5) Affect
(6) Weight
(7) Skin
(8) Hair
(9) Personal appearance
(10) Sexual function
i. Specific history of
(1) Hypopituitarism
(2) Hypothyroidism
(3) Polydipsia
(4) Polyuria
(5) Polyphagia
(6) Diabetes
(7) Exophthalmus in hyperthyroidism

4. Focused physical examination
a. Appearance
b. Level of consciousness
c. Apparent state of health
d. Skin color
e. Vital signs

C. Management/ treatment plan
1. Airway and ventilatory support
a. Maintain an open airway

b. High flow oxygen
2. Circulatory support
   a. Monitor blood pressure
3. Pharmacological interventions
   a. Consider initiating intravenous line
   b. Avoid interventions which mask signs and symptoms
4. Non-pharmacological interventions
   a. Monitor LOC
   b. Monitor vital signs
5. Transport consideration
   a. Appropriate mode
   b. Appropriate facility
6. Psychological support
   a. All actions reflect a calm, caring, competent attitude
   b. Keep patient and significant others informed of your actions

III. Specific illnesses
A. Diabetes mellitus
1. Epidemiology
   a. Incidence
   b. Morbidity/ mortality
   c. Long term complications
   d. Risk factors
   e. Prevention strategies
2. Anatomy and physiology review
3. Pathophysiology
   a. Types
      (1) Type I-insulin dependent
      (2) Type II-non insulin dependent
   b. A chronic system syndrome characterized by hyperglycemia caused by a decrease in the secretion or activity of insulin
   c. Normal insulin metabolism
      (1) Produced by beta cells in the islets of Langerhans
      (2) Continuously released into the bloodstream
         (a) Insulin is released from the beta cells as proinsulin
         (b) Routed through the liver where 50-70 percent is extracted from the blood
         (c) The level of plasma insulin rises after a meal
            i) Stimulates storage of glucose as glycogen, liver and muscle tissue
            ii) Enhances fat deposition in adipose tissue
            iii) Inhibits protein degradation
            iv) Accelerates protein synthesis
         (d) The fall of plasma insulin levels during normal overnight fasting facilitates the release of
            i) Stored glucose from the liver
            ii) Protein from muscle tissue
            iii) Fat from adipose tissue
         (e) Average daily secretion is 0.6 units per kilogram of body weight
      (3) Activity of released insulin

(a) Lowers blood glucose levels
(b) Facilitates a stable, normal glucose range of approximately 70 to 120 mg/ dl

d. Ketone formation
(1) When insulin supply is insufficient, glucose cannot be used for cellular energy
(2) Response to cellular starvation
(3) Body releases and breaks down stored fats and protein to provide energy
(4) Free fatty acids from stored triglycerides are released and metabolized in the liver in such large quantities that ketones are formed
(5) Excess ketones upset the pH balance and acidosis develops
(6) Gluconeogenesis from protein is the last source used by the body as a compensatory response to provide cellular energy
(a) Results in an increase in glucose and nitrogen
(b) Due to prevailing insulin insufficiency, the glucose can not be used resulting in
i) Increased osmotic diuresis
ii) Dehydration and loss of electrolytes, particularly potassium

4. Assessment findings
a. History
(1) Has insulin dosage changed recently?
(2) Has the patient had a recent infection?
(3) Has the patient suffered any psychologic stress?
b. Signs and symptoms
(1) Altered mental status
(2) Abnormal respiratory pattern (Kussmaul's breathing)
(3) Tachycardia
(4) Hypotension
(5) Breath has a distinct fruity odor
(6) Polydipsia
(7) Polyphagia
(8) Warm dry skin
(9) Weight loss
(10) Weakness
(11) Dehydration

5. Management
a. Airway and ventilation
b. Circulation
c. Pharmacological interventions
d. Non-pharmacological interventions
e. Transport consideration
(1) Appropriate mode
(2) Appropriate facility
f. Psychological support/ communication strategies

B. Hypoglycemia
1. Epidemiology
a. Incidence
b. Morbidity/ mortality
c. Risk factors

d. Prevention strategies

2. Pathophysiology
   a. Blood glucose levels fall below that required for normal body functioning
   b. Combined effects of a decreased energy supply to the central nervous system and a hyperadrenergic state results from a compensatory increase in catecholamine secretion
      (1) Tremors
      (2) Diaphoresis
      (3) Palpitations
      (4) Tachycardia
      (5) Pale, cool skin
      (6) Low levels of blood glucose reaching the brain results in an altered mental status
      (7) Irritability
      (8) Confusion
      (9) Stupor
      (10) Coma
3. Assessment
   a. Known history of
      (1) Diabetes
      (2) Prolonged fasting
      (3) Alcoholism
   b. Signs and symptoms
      (1) Weakness
      (2) Irritability
      (3) Hunger
      (4) Confusion
      (5) Anxiety
      (6) Bizarre behavior
      (7) Tachycardia
      (8) Normal respiratory pattern
      (9) Cool, pale skin
      (10) Diaphoresis
4. Management
   a. Airway and ventilation
   b. Circulation
   c. Pharmacological interventions
   d. Non-pharmacological interventions
   e. Transport consideration
      (1) Appropriate mode
      (2) Appropriate facility
      (3) Psychological support/ communication strategies

C. Hyperglycemia (hyperglycemic hyperosmolar nonketosis)
1. Epidemiology
   a. Incidence
   b. Mortality/ morbidity
   c. Risk factors
   d. Prevention strategies
2. Pathophysiology
   a. Occurs in patients with diabetes who are able to produce enough insulin to

prevent DKA but not enough to prevent severe hyperglycemia, osmotic diuresis and extracellular fluid depletion
b. Increasing blood glucose levels causes a fluid shift from intracellular to extracellular spaces

3. Assessment
   a. Known history of
      (1) Diabetes
      (2) Inadequate fluid intake
   b. Signs and symptoms
      (1) Neurologic abnormalities
         (a) Somnolence
         (b) Coma
         (c) Seizures
         (d) Hemiparesis
         (e) Aphasia
         (f) Increasing mental depression
         (g) Dehydration
         (h) Polydipsia
         (i) Polyuria
         (j) Polyphagia
4. Management
   a. Airway and ventilatory support
   b. Circulation
   c. Pharmacological interventions
   d. Non-pharmacological interventions
   e. Transport consideration
      (1) Appropriate mode
      (2) Appropriate facility
   f. Psychological support/ communication strategies

D. Diabetic ketoacidosis
1. Epidemiology
   a. Incidence
   b. Mortality/ morbidity
   c. Risk factors
   d. Prevention strategies
   e. Anatomy and physiology review
2. Pathophysiology
   a. Hyperglycemia
   b. Ketonemia
   c. Relative insulin insufficiency
   d. Counterregulatory hormone excess
3. Assessment findings
   a. History
      (1) General health
      (2) Previous medical conditions
      (3) Medications
      (4) Previous experience with complaint
      (5) Time of onset
   b. Physical
      (1) Dehydration

(2) Hypotension
(3) Reflex tachycardia
(4) Acetone (fruity) odor on breath
(5) Nausea
(6) Vomiting
(7) Abdominal pain
(8) Hyperventilation
(9) Kussmall's respiration

4. Management
   a. Airway and ventilatory support
      (1) Oxygen
      (2) Positioning
      (3) Suction
      (4) Assisted ventilation
      (5) Suction
      (6) Advanced airway devices
   b. Circulatory support
      (1) Venous access
      (2) Blood analysis
   c. Non-pharmacological interventions
      (1) General comfort measures
   d. Pharmacological interventions
      (1) Rehydration
      (2) Bicarbonate
      (3) Potassium
      (4) Insulin
   e. Psychological support
   f. Transport considerations
      (1) Appropriate mode
      (2) Appropriate facility

E. Thyrotoxicosis (thyroid storm)
   1. Epidemiology
      a. Incidence
      b. Mortality/ morbidity
      c. Risk factors
      d. Prevention strategies
   2. Pathophysiology
      a. Acute manifestation of all hyperthyroid symptoms
      b. Excessive circulating level of thyroxine and triiodothyronine
         (1) Regulate metabolism
         (2) Regulate growth and development
   3. Assessment
      a. History
      b. Signs and symptoms
         (1) Severe tachycardia
         (2) Heart failure
         (3) Cardiac dysrhythmias
         (4) Shock
         (5) Hyperthermia
         (6) Restlessness

(7) Agitation
(8) Abdominal pain
(9) Delirium
(10) Coma

4. Management
   a. Airway and ventilation
   b. Circulation
   c. Pharmacological interventions
      (1) Anti-thyroid drugs - in hospital management
      (2) Beta adrenergic receptor blockers
   d. Non-pharmacological interventions
   e. Transport consideration
      (1) Appropriate mode
      (2) Appropriate facility
   f. Psychological support/ communication strategies

F. Myxedema (adult hypothyroidism)
   1. Epidemiology
      a. Incidence
      b. Mortality/ morbidity
      c. Risk factors
      d. Prevention strategies
   2. Pathophysiology
      a. A disease caused by hyposecretion of the thyroid gland during the adult years
   3. Assessment
      a. History
      b. Signs and symptoms
         (1) Edematous face
         (2) Periorbital edema
         (3) Mask-like effect
         (4) Impaired memory
         (5) Slowed speech
         (6) Decreased initiative
         (7) Somnolence
         (8) Cold intolerance
         (9) Dry, coarse skin
         (10) Muscle weakness and swelling
         (11) Constipation
         (12) Weight gain
         (13) Hair loss
         (14) Hoarseness
   4. Management
      a. Airway and ventilation
      b. Circulation
      c. Pharmacological interventions
      d. Non-pharmacological interventions
      e. Transport consideration
         (1) Appropriate mode
         (2) Appropriate facility
      f. Psychological support/ communication strategies

IV. Corticosteroid excess - Cushing's syndrome

A. Epidemiology
   1. Incidence
   2. Mortality/ morbidity
   3. Risk factors
   4. Prevention strategies

B. Pathophysiology
   1. A spectrum of clinical abnormalities caused by an excess of corticosteroids, especially glucocorticoids
   2. Causes
      a. Corticotropin secreting pituitary tumor
      b. Cortical secreting neoplasm within the adrenal cortex
      c. Excess secretion of corticotropin by a malignant growth outside the adrenal
      d. Prolongs administration of high dose corticosteroids

C. Assessment
   1. History
   2. Signs and symptoms
      a. Thinning hair
      b. Acnes
      c. Hump on back of neck (buffalo hump)
      d. Supraclavicular fat pad
      e. Thin extremities
      f. Ecchymosis
      g. Slow healing
      h. Pendulous abdomen
      i. Weight gain
      j. Increased body and facial hair

D. Management
   1. Airway and ventilation
   2. Circulation
   3. Pharmacological interventions
   4. Non-pharmacological interventions
   5. Transport consideration
      a. Appropriate mode
      b. Appropriate facility
   6. Psychological support/ communication strategies

V. Adrenal insufficiency - Addison's disease

A. Epidemiology
   1. Incidence
   2. Mortality/ morbidity
   3. Risk factors
   4. Prevention strategies

B. Pathophysiology
   1. Adrenal insufficiency
      a. Adrenal steroids are reduced
         (1) Glucocorticoids
         (2) Mineralocorticoids
         (3) Androgens
   2. Most common cause is idiopathic atrophy of adrenal tissue
   3. Less common caused include hemorrhage, infarctions, fungal infections and acquired

immune deficiency disease

C. Assessment
   1. History
   2. Signs and symptoms
      a. Progressive weakness
      b. Progressive weight loss
      c. Progressive anorexia
      d. Skin hyperpigmentation
         (1) Areas exposed to the sun
         (2) Areas exposed to pressure points
         (3) Joints and creases
      e. Hypotension
      f. Hyponatremia
      g. Hyperkalemia
      h. Nausea
      i. Vomiting
      j. Diarrhea

D. Management
   1. Airway and ventilation
   2. Circulation
   3. Pharmacological interventions
   4. Non-pharmacological interventions
   5. Transport consideration
      a. Appropriate mode
      b. Appropriate facility
   6. Psychological support/ communication strategies

VI. Integration